## A Case of Asthma COPD Overlap

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Received: March 2019 Accepted: March 2019

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#### **ABSTRACT**

Asthma and COPD are the pulmonary diseases most frequently encountered in clinical practice. Usually, each disease is caused by a different etiology and shows a different clinical picture and course. However, these two diseases sometime present within the same patient, and it is now recognized that asthma and COPD can coexist as asthma COPD overlap (ACO), which is clinically important for several reasons. First, it is estimated that the number of patients with ACO will increase significantly together with the recent increase in numbers of patients with asthma and COPD. Secondly, patients with ACO are prone to experience more frequent and severe exacerbations. Patients who have asthma with a COPD component tend to present with severe hypoxia because of Irreversible/fixed airway obstruction and impairment of the alveolar diffusion capacity by emphysematous changes. In contrast, patients with COPD who have an asthma component not only have exertional dyspnea but also develop paroxysmal wheezing or dyspnea at night or in the early morning. Here we report a case of 60 yr old male diagnosed as a case of asthma COPD overlap.

Keywords: Asthma, COPD, Asthma COPD overlap.

#### INTRODUCTION

Bronchial asthma (asthma) is defined as follows: "Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness, and cough that vary over time and in intensity, together with variable expiratory airflow limitation".[1] And chronic obstructive pulmonary disease (COPD) is defined as follows: "Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases".[2] Asthma and COPD appear as a result of different mechanisms triggered by different pathogeneses and although they present different features and symptoms of airway inflammation and airway obstruction, there are also cases that present the features of both asthma and

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COPD. This type of pathology is known as asthma-COPD overlap syndrome (ACOS). In 2014 the GINA &GOLD Joint Committee defined it as follows: "Asthma-COPD overlap syndrome (ACOS) is characterized by persistent airflow limitation with several features usually associated with asthma and several features usually associated with COPD. ACOS is therefore identified by the features that it shares with both asthma and COPD".[3] However, subsequently for a variety of reasons the term "syndrome" a term that is normally used to refer to an idiopathic (in some cases, the term continues to be used even after the cause is identified) pathology with features in common (e.g. clinical symptoms, test findings) came to be considered inappropriate in this context. Those reasons included the fact that the pathogeneses of asthma and COPD are not a single phenomenon but rather are formed due to a variety of mechanisms and the fact that the clinical features are highly diverse. As a result, it has been recommended that the term "syndrome" be dropped and that the disease name be changed to "asthma and COPD overlap (ACO)". The disease then was described as follows: "Asthma-COPD overlap (ACO) is characterized by persistent airflow limitation with several features usually associated with asthma and several features usually associated with COPD. Asthma-COPD overlap is therefore identified in clinical practice by the features that it

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shares with both asthma and COPD. This is not a definition, but a description for clinical use, as asthma-COPD overlap includes several different clinical phenotypes andthere are likely to be several different underlying mechanisms". In this paper, the disease that shares several features of both asthma and COPD will be referred to as ACO.

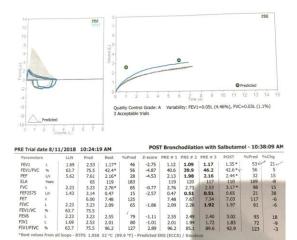
#### **CASE REPORT**

A 60 yr male presented with fever, cough with expectoration for the last 2 weeks and breathlessness since 2 days. He gives history of breathlessness since childhood from 15 years of age. There is history of seasonal variation of breathlessness with symptoms on exposure to dust and was more during cold climate that often needs hospitalisation. He gives history smoking for the past 45 years with more than 10 pack years. There is history of exertional dyspnoea for the last 4 years which makes it difficult even during day to day activities. On examination patient was dyspnoeic, with a blood pressure 130/90,pule rate 108/minute, respiratory rate 30/minute and saturation was 86% on room air. On general physical examination pallor and clubbing present. Respiratory examination revealed bilateral ronchi with decreased breath sounds.

Laboratory findings showed a white cell count of 14600, Haemoglobin 13.3g/dl, ESR 20mm/hr, RBS 112mg/dl, Serum Bilirubin 0.54,SGOT 31,SGPT 26,S ALP 0.90. His serum electrolytes and renal function test were within normal ranges. His sputum examination for acid fast bacilli and fungus were negative, sputum for gram stain and culture and sensitivity showed growth of pseudomonas, sputum for CBNAAT MTB not detected, chest xray showed bilateral hyperinflation and flattening of diaphragm.



Spirometry showed FEV1 46 % Post Bronchodilator FEV1 53 % FEV1/FVC 56% Post Bronchodilator FEV1/FVC 56% Post Bronchodilator Increase in FEV1 21%



#### DISCUSSION

The differentiation in terms of respiratory symptoms between asthma and COPD is, in many cases, quite difficult, because there are several areas where they overlap, making the distinction more complicated. For example, the presence of chronic productive cough is more associated with COPD but can also be present in an asthmatic patient, which leads to a worse prognosis in terms of pulmonary function decline.<sup>[4]</sup> On the other hand, it is also common to have the presence of asthmatic symptoms (occasional dyspnoea, sibilance) in COPD patients.<sup>[5]</sup> In terms of bronchodilator response, the reversibility seen, although typical in asthma, is not exclusive to it, as it is also observed in up to 50% of COPD patients. [6] Furthermore, the bronchial hyper responsiveness, which is present in almost all asthma patients, can also be seen in a significant percentage of COPD cases.<sup>[7]</sup> This clinical diversity leads to the overlap between these two obstructive respiratory diseases. It is possible, how-ever, to isolate characteristics that enable the recognition of a new entity (ACO) which aggregates features from both (asthma and COPD).

In relation to radiological differences between ACO and COPD, there seems to be a slightly less emphysema expression in the first group as well as a predilection for the upper lung lobes.<sup>[8]</sup>

There is a higher degree of consensus between the studies over exacerbations, with a higher rate in ACO patients. [9-11] There is also a higher percentage of severe exacerbations and a higher rate of hospitalizations. In a study performed by Brzostek and Kokota significant rate of recent exacerbations (69% in the last year) was observed. [12] In terms of comorbidities, studies point towards a high prevalence in ACO, especially cardiovascular ones. Some authors have referred to a higher incidence in ACO patients compared to COPD and asthma but that is not applicable to the whole literature. [12-14]

The diagnosis of ACO should be considered in the concomitant presence of:1) simultaneous clinical manifestations characteristic of both asthma and

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COPD2) persistent airway obstruction, defined as post-bronchodilator FEV1/FVC < 0.7, evaluated in a period of clinical stability3) positive response in bronchodilator test, defined by an increase in the value of FEV1of ≥200 mL and ≥12% frombaseline4) current or past history of smoking or exposure to biomass combustion As aspects that are usually present in this group of patients and that can be taken into account in the diagnostic consideration, we highlight peripheral eosinophilia (>300 eosinophils/\_L or >5% of leukocytes) and previous his-tory of atopy.

ICS/LABA as first line therapy. In patients which are not controlled or whose clinical severity justifies, a triple therapy with ICS/LABA/LAMA should be used.- Non-pharmacological therapy such as pulmonary rehabilitation should be done in ACO patients with uncontrolled symptomatology (frequent exacerbations).- Comorbidities treatment should be optimized for a better control of the lung disease.- Risk factors exposure control (smoking, biomass, allergens exposure) and vaccination coverage (influenza and anti-pneumococcal).

# In the Above Reported Case, the Following Points are in the Favour of Diagnosis of ACO

- 1. Usually age ≥40 years, but may have had symptoms in childhood or early adulthood
- 2. Respiratory symptoms including exertional dyspnea are persistent but variability may be prominent
- 3. Airflow limitation not fully reversible, but often with current or historical variability
- 4. Persistent airflow limitation
- 5. Frequently a history of doctor- diagnosed asthma (current or previous), allergies and a family history of asthma, and/or a history of noxious exposures
- Symptoms are partly but significantly reduced by treatment. Progression is usual and treatment needs are high
- 7. Exacerbations may be more common than in COPD but are reduced by treatment.
- 8. Comorbidities can contribute to Impairment
- 9. Post-BD FEV1/FVC < 0.7
- 10. Post-BD FEV1<80% predicted
- 11. Post-BD increase in FEV1>12%

#### **CONCLUSION**

The present study sets forward the heterogeneity of diagnosis that still exists in this area, which underlines its importance as a first stage in the examination of this field. It seems clear there is a group of patients who share characteristics that cross the COPD and asthma spectrum, it is therefore crucial to achieve a more accurate identification of these patients, enabling a more effective therapeutic approach. In the future this characterization of ACO patients will provide for the development of national prevalence studies and the evaluation of the impact of different pharmacological and non-

pharmacological therapies, which will complement our knowledge of this entity and optimize treatment strategies.

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How to cite this article: Mathew L, Kajal NC, Mahajan S, Nithin KT, Sharma S, Neki NS, Sharma M. A Case of Asthma COPD Overlap. Ann. Int. Med. Den. Res. 2019; 5(3):TB01-TB03.

Source of Support: Nil, Conflict of Interest: None declared